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Fulminant Acute Colitis following a Self-Administered Hydrofluoric Acid Enema

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A 33-yr-old white male presented with bloody diarrhea, leukocytosis, and left lower quadrant direct and rebound tenderness after a self-administered concentrated hydrofluoric acid enema while intoxicated from intranasal cocaine administration. Intraoperative flexible sigmoidoscopy and a gastrografin enema revealed severe mucosal ulceration and edema in the rectum and sigmoid colon. Laparotomy revealed an ulcerated, necrotic, and purulent sigmoid colon and intraperitoneal pus. The patient underwent a limited sigmoid resection and a Hartman procedure. Five months later, the patient presented with a rectal stricture which was resected. This case demonstrates that a hydrofluoric acid enema can cause fulminant acute colitis and chronic colonic strictures.

INTRODUCTION

Although the clinical presentation of upper gastrointestinal injury from acid ingestion has been well described (1–5), little is known about the clinical presentation of lower gastrointestinal injury from an acid edema (6, 7). We report the clinical, radiographic, endoscopic, and pathologic findings in a case of hydrofluoric acid administered by enema.

CASE REPORT

A 33-yr-old previously healthy, human immunodeficiency virus seronegative white male, with a history of chronic intranasal cocaine abuse, presented with severe rectal pain and bloody diarrhea 36 h after self-administering a concentrated hydrofluoric acid enema while intoxicated from intranasal administration of cocaine. He denied a history of homosexuality, rectal trauma, or anal intercourse. Physical examination revealed an alert male in pain. The temperature was 36.7°C, the pulse was 86 beats/min, and the blood pressure was 150/90 mm Hg. Lung and heart examinations were within normal limits. There was left lower quadrant direct and rebound tenderness with localized guarding. Bowel sounds were hypoactive. The perianal skin was excoriated. Rectal examination revealed an exquisitely tender anus and bloody stool. The hematocrit was 44.9. There were 15,300 leukocytes/mm^3, of which 62% were neutrophils, and 24% were band forms. The serum calcium level was 8.2 mg/dl (normal., 8.6–10.4), with a normal serum albumin level. Values of other routine serum electrolytes and biochemical parameters of liver function were within normal limits. The platelet count, prothrombin time, and partial thromboplastin time were within normal limits. Abdominal roentgenogram revealed no subdiaphragmatic "free air."

The patient received calcium carbonate enemas to bind intraluminal fluoride ion, and iv administered ampicillin, gentamicin, and clindamycin. A gastrografin enema revealed mural spiculations due to ulceration, thumbprinting due to submucosal edema, a widened presacral space due to retrorectal inflammation, bowel angulation, subtotal obstruction in the distal descending colon, and no extravasation of contrast (Fig. 1). Sigmoidoscopy performed in the operating room, after induction of general anesthesia, revealed severely ulcerated and necrotic rectal and sigmoid mucosa. An exploratory laparotomy was performed which revealed a discolored, purulent sigmoid and intraperitoneal pus. Fourteen centimeters of sigmoid were resected, and a Hartman procedure with a distal sigmoid colostomy was performed. Bilateral ureteral cathethers and percutaneous perirectal drains were inserted. Pathologic examination of the resected specimen revealed profound ulceration, necrosis, acute inflammation, and submucosal bacterial proliferation (Figs. 2 and 3). There was no mesenteric thrombus, embolus, or atherosclerosis. The patient was discharged 14 days later after an uneventful postoperative recovery.

The patient was readmitted electively 5 months later for bowel reanastomosis. The patient denied cocaine use or toxic enema insertion since the prior hospitalization. Sigmoidoscopy revealed a friable mucosa, rigid bowel wall, and a stricture 10 cm from the anal verge beyond which it was not possible to pass. At laparotomy, the stricture was resected and the bowel was

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reanastomosed. Pathologic examination of the resected specimen revealed mucosal erosions and congestion, chronic inflammation, and dense fibrosis. The patient was discharged 8 days after surgery.

DISCUSSION

Hydrofluoric acid

Hydrofluoric acid is a colorless liquid manufactured from sulfuric acid and calcium fluoride. It is used commercially to remove rust in semiconductor manufacture, to catalyze petrochemical reactions, and to etch glass (8). Injury may occur by skin contact, ocular exposure, ingestion, or inhalation. Direct contact causes liquefaction necrosis of soft tissues. Local penetration causes decalcification and erosion of bone by fluoride binding to calcium (9). Systemic absorption can cause hypocalcemia and consequent arrhythmias because of fluoride binding to serum calcium. It can also cause metabolic acidosis, hyperkalemia, hypomagnesemia, acute pulmonary edema, and myocardial injury (10).

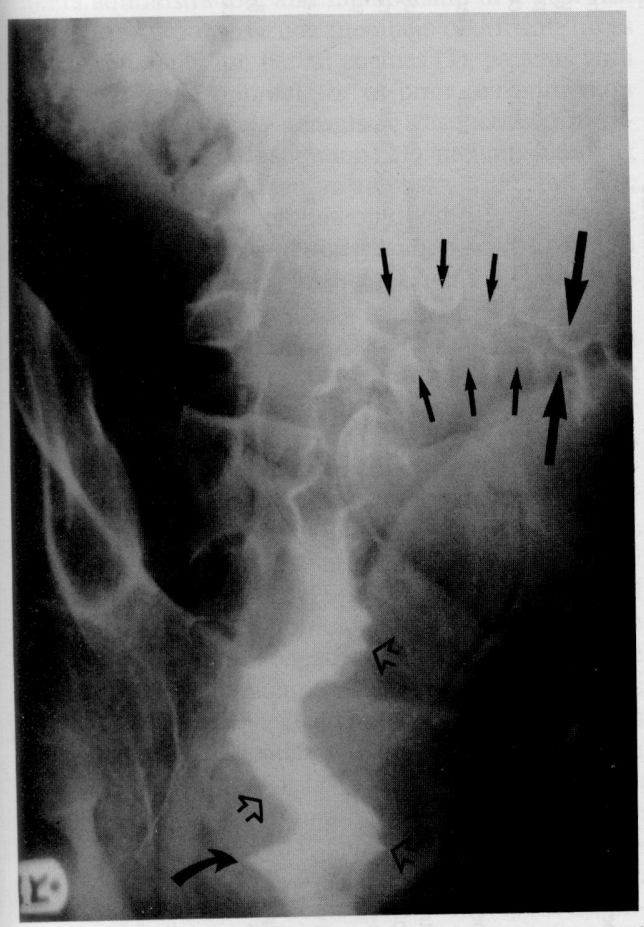

FIG. 1. Oblique view of a gastrografin enema reveals massive mural thumbprinting due to submucosal edema (*small arrows*), mural spiculations due to mucosal ulceration (*open arrows*), bowel angulation (*curved arrow*), and marked luminal narrowing with subtotal obstruction at the descending-sigmoid junction (*large arrows*).

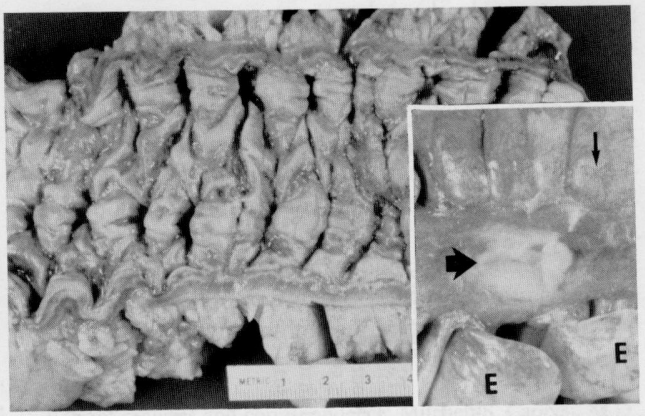

FIG. 2. Gross pathologic view of the luminal aspect of the resected sigmoid colon reveals cobblestoning of the sigmoid mucosa due to profound linear ulcers. There is also diffuse mucosal erythema, focal hemorrhage, and mural thickening. *Inset* shows gross pathologic view of the serosal aspect of the resected specimen. Note the edematous colonic folds (labeled *E*), serosal vascular congestion and erythema (*slender arrows*), and focal transmural inflammation (*wide arrow*).

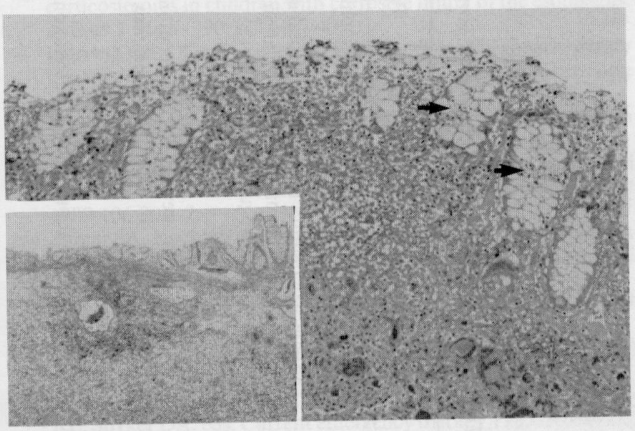

FIG. 3. High power photomicrograph from the resected sigmoid from an area near an ulcer. The colonic glands are widely separated due to a massive acute and chronic inflammatory infiltrate. Inflammatory cells invade two colonic glands (*arrows*) (hematoxylin and eosin, ×200). *Inset* shows a low power photomicrograph from the edge of a profound sigmoid ulcer. The normal mucosal and submucosal architecture is disrupted and replaced with a fibrinopurulent inflammatory exudate and granulation tissue, except for a few preserved colonic glands at the right edge of the ulcer (hematoxylin and eosin, ×40).

Ingestion can cause nausea, vomiting, abdominal pain, diarrhea, and a hemorrhagic esophagitis and gastritis (10–12). Emetics are contraindicated because this acid is caustic. Gastric lavage should be performed within 90 min of ingestion if spontaneous emesis has not occurred. Calcium gluconate is preferred for gastric lavage because calcium can bind unabsorbed fluoride (10). The currently reported patient similarly received calcium carbonate enemas to bind unabsorbed fluoride in the colon. Skin exposed during toxic ingestion or toxic enema insertion should be treated with topical

TABLE 1
Previously Reported Colonic Toxicity due to Intrarectally Administered Chemical or Medicinal Agents

Medication or Chemical	No. of Cases	Clinical Findings	Endoscopic & Radiographic Findings	Pathology	Suspected Pathophysiology	References
Chloro-m-xylenol (dettol)	1	Bloody diarrhea	Mucosal edema, granularity, friability	Neutrophilic infiltrate	Moderate toxin at >5% concentration	7
Detergent enemas	7	Bloody diarrhea, abdominal pain, vomiting, pyrexia, abdominal tenderness, abdominal distention, leukocytosis	Friable, edematous, granular, and erythematous mucosa, serosanguinous exudate	Mucosal edema, capillary dilation	Irritation from high soap concentration	7, 21–26
Ergotamine	3	Abdominal pain, hematochezia, mucoid diarrhea, tenesmus, leukocytosis	Anorectal ulcers, stenosis	Ulceration, inflammation, necrosis	Local vasospasm	14–15
Ethyl alcohol	1	Abdominal pain, hematochezia	Erythema, ulceration	Ulceration, inflammation	Moderate topical toxin when 100% pure	27
Herbal medicines	5	Rectal bleeding, pyrexia, abdominal pain, perianal excoriation	Distended colon proximal to a stricture	Hemorrhagic necrosis, inflammation, ulceration	Various toxins	7
Hydrogen peroxide	34	Bloody diarrhea, abdominal pain, pyrexia, tenesmus, leukocytosis	Friability, granularity, stricture, ulceration, purulent exudate, edema, pneumatosis coli, frothy colonic fluid	Fibrinopurulent exudate, ulcers, inflammation, pneumatosis coli, mucosal hemorrhage	Intracellular explosive oxygen production	18, 19, 28–30
Potassium permanganate	1	Bloody diarrhea	Mucosal friability & hemorrhage	Hemorrhagic colits	Corrosive oxidizing agent	7
Sodium diatrizoate, meglumine (hypaque, renografin-76)	2	Pyrexia, abdominal pain & tenderness, abdominal distention	Friable mucosa, dilated lumen, retained colonic contrast	Mucosal edema, inflammation & necrosis, thin muscularis mucosa	Prolonged colonic exposure with hypertonic agent in obstructed colon	16, 17
Acetic acid (vinegar)	1	Rectal bleeding, pyrexia	Intraperitoneal "free air"	Bowel infarction	Corrosive at very high concentrations	7
Sodium hydroxide (caustic soda)	1	Abdominal pain & tenderness, rectal tenderness, rectal bleeding, pyrexia, leukocytosis	Purulent exudate, hemorrhage, ulcers, luminal narrowing	Submucosal fibrosis, mucosal inflammation	Corrosive agent	20

application of calcium gluconate to bind fluoride ion (8, 10, 13).

Colonic toxicity of intrarectally administered agents

Colitis has been reported due to intrarectally administered medications, illicit drugs, contrast materials, detergents, and powerful acids, bases, or other corrosive substances (Table 1). Toxins are introduced per rectum by suppository, enema, or colonoscopic fluid instillation. Toxic exposure may result from conventional medical therapy (14, 15), unconventional medical therapy (7), radiographic examination (16, 17), colonoscopic examination (18, 19), deliberate self-mutilation (20), or accidental self-administration (current report).

Although a toxic colitis may be obvious from the history, this etiology may not be recognized in an intoxicated patient, a malingering patient, or a patient unaware of the importance of relating this history. Clues that a colitis may be due to an intrarectally administered agent include perianal excoriation, a segmental distal colitis due to a concentration gradient from enema administration, and membership in a high risk group. Risk groups include members of African tribes who traditionally use herbal enemas (7), patients with a history of schizophrenia or of prior self-mutilation (20), patients who self-administer unsupervised medications for chronic constipation (21), patients receiving a hypertonic contrast agent in an obstructed colon (16), patients undergoing colonoscopy with a colonoscope inadequately washed after sterilization with hydrogen peroxide (18, 19), and patients receiving high-concentration detergent enemas (22).

The reported clinical, endoscopic, radiographic, and pathologic findings with toxins introduced per rectum are listed in Table 1 (6, 7, 14–30). The clinical presentation depends upon the toxin, toxic concentration, exposure duration, route of exposure, time after exposure, general medical status, and presence of prior colonic disease. Clinical findings with caustic colitis include fever, abdominal pain, nonbloody or bloody diarrhea, excretion of mucopus, tenesmus, abdominal tenderness, rectal tenderness, and leukocytosis (Table 1). Although the clinicopathologic findings in caustic esophagitis are well documented (1–5), only a few cases of corrosive colitis have been reported (Table 1). This is due to the much greater frequency of caustic ingestion than caustic enema. Caustic ingestion initially produces cellular edema, inflammation, and necrosis in the esophagus (3, 5, 21). Esophageal ulceration subsequently develops from sloughing of necrotic mucosa. Granulation tissue and fibrosis form with repair. Esophageal complications include perforation and hemorrhage from acute injury, and obstruction due to cicatrization with chronic injury (2–5, 21).

The currently reported patient had also taken cocaine, but did not present with an ischemic colitis as expected, with mesenteric vasoconstriction caused by cocaine (31–33). Rather, this patient developed a fulminant segmental distal colitis as expected from a corrosive enema. This patient developed acute fulminant mucosal ulceration, gastrointestinal bleeding, impending colonic perforation, and a subsequent chronic stricture. The chronic stricture did not produce symptoms because the stricture was in the distal excluded colonic segment. The reported colonic injury produced by an acid enema resembles the esophageal injury produced by acid ingestion.

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